An Outline of the Diseases

of the

AVIAN LEUKOSIS COMPLEX

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AVIAN LEUKOSIS COMPLEX

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INTRODUCTION

An attempt has been made to summarize in one table the current information concerning the various diseases of the avian leukosis complex. It now appears that at least two groups of viruses or agents are responsible for these diseases. Outstanding distinguishing characteristics are (1) whether or not the virus grows in chicken embryo fibroblast and induces resistance and (2) whether or not the virus causes the formation of a group specific complement fixing antigen. The cause of the induced resistance was referred to by Rubin (1960, 1961) [2] as RIF, an acronym for resistance-inducing factor, and later determined to be naturally occurring leukosis virus. Sarma and others (1964) used hamster anti-Rous sarcoma (Schmidt-Ruppin strain) serum to detect an avian leukosis group antigen. This test has been called COFAL, an acronym for complement fixation avian leukosis. The RIF and COFAL positive viruses cause lymphoid leukosis (formerly included under visceral lymphomatosis), myeloblastosis, erythroblastosis, fibrosarcoma, endothelioma, nephroblastoma, and probably osteopetrosis. Recent data by Vogt (1965 a and b) and Hanafusa (1965) indicate that the RIF-positive leukosis viruses occur in three antigenically distinct subgroups, A, B, and C. Viruses of each subgroup have a corresponding type of Rous sarcoma virus (RSV) to which they act as specific resistance-inducing factors. Also, susceptibility to infection is determined by single autosomal genes specific for each virus subgroup (Crittenden and others (1964)).

The agents that do not grow in tissue culture hence do not induce resistance, are referred to here as RIF-negative. These agents are also COFAL-negative and cause Marek's disease (also formerly included under the term "lymphomatosis") with lesions of the nerves, viscera, muscle, skin, and eyes (Biggs and Payne 1964). The visceral lesions are distinguishable with difficulty from those caused by the RIF-positive leukosis virus.

The classification and nomenclature proposed by Biggs (1961, 1963) which take into account etiology and epizootiology as well as pathology are here employed. The term "lymphomatosis" has formerly been used for all lesions of similar morphology irrespective of etiologic differences. It is not employed in this classification, to avoid confusion associated with its use in diseases of different etiology. In its place two terms are employed—"lymphoid leukosis" for the lymphoid proliferations caused by the RIF-positive viruses and "Marek's disease" for all the proliferative lymphoid lesions of the nervous system including the oculi, the viscera, the skeletal muscle, and the skin, caused by RIF-negative agents.

The term "acute leukosis" has been applied to outbreaks of neural and visceral leukosis in young birds. Skin and muscle leukosis has been reported to occur in broiler flocks. The etiology of these diseases is still largely unknown. Because of the similarity in epizootiology and pathology of these disease syndromes to those caused by known RIF-negative leukosis agent, they have been included here under the description of Marek's disease.

It is quite obvious that much more critical information will have to be obtained concerning several aspects of the diseases of this complex before they can be adequately understood and a satisfactory classification agreed upon.

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[2] Names followed by year in parentheses refer to Literature Cited.

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THE AVIAN LEUKOSIS COMPLEX

Item	Diseases Caused by RIF-Positive Viruses						Diseases Caused by RIF-Negative Agents
	Lymphoid Leukosis (LL)	Myeloblastosis (Leukemic)	Myelocytomatosis (Aleukemic)	Erythroblastosis		Fibrosarcoma, endothelioma, and nephroblastoma	Osteopetrosis	Marek's disease (MD) (including acute leukosis)
				Proliferative	Anemic			
Synonyms	Visceral lymphomatosis, lymphocytoma, big liver disease.	Cranuloblastosis, myeloleukosis, diffuse myeloid leukosis.	Leukochloroma, myelocytoma, discrete myeloid leukosis, erythroleukosis.	Intravascular lymphoid leukosis.	Erythroleukosis, erythromyelosis.	----------	Marble bone, thick leg disease.	Neural lymphomatosis, fowl paralysis, range paralysis, neuritis. Visceral lymphomatosis, acute leukosis. Ocular lymphomatosis, gray eye, pearly eye, iritis.
Definition	Autonomous proliferation of the respective immature blood elements with impairment of organ function.					Autonomous proliferation of fibroblasts, vascular endothelial, renal epithelial, and other cells.	Excessive proliferation of bone cells and deposition of hard bone.	Infiltration of lymphocytes and plasma cells with progression to autonomous proliferation.
History	Caprini, 1896, 1st to describe; Ellermann and Bang, 1908, transmitted with filtrates and proved viral etiology of erythroblastosis; Burmester and others, 1946, showed viral etiology of lymphoid leukosis and relation to erythroblastosis.					Rous, 1909, transmitted fibrosarcoma with virus; Furth, 1933, transmitted endothelioma; Carr, 1956, transmitted renal adenocarcinoma.	Pugh, 1927, described diffuse osteopetrosis; Jungherr, 1935, transmitted and indicated relation to lymphomatosis.	Marek, 1907, and Kaupp, 1921, described the disease, and the latter associated blindness with paralysis. Vander Walls and Winkler-Junius, 1924, and Pappenheimer, Dunn, and Cone, 1926, described and transmitted the disease. The latter authors noted the common occurrence of tumors of the viscera, especially of the ovary.
Etiology: Size, shape, and structure.	A family of closely related viruses, ovoid or spherical in shape; about 800 A in diameter with an electron dense core.					Differences are indistinguishable.	Specific virus not yet isolated.	Morphology of specific agent has not been described.
RIF activity	All viruses tested are RIF-positive, that is, induce resistance to foci formation by Rous sarcoma virus (RSV) types A, B, or C.					Rous associated viruses (RAV) are RIF-positive.	Most RIF-positive viruses cause osteopetrosis.	All strains (JM and B14) tested lack RIF activity.
COFAL	All RIF-positive viruses are also COFAL-positive.							All strains tested are COFAL-negative.
Viability	Killed by common disinfectants. Activity lost rapidly at room temperature but remains viable for long periods at −76° C.							Viability is maintained for moderate periods at room temperature, but is reduced by freezing.
Tissue culture	Grows well in susceptible cultures of avian tissue. Some strains of Rous sarcoma virus will produce tumors in mammals and a transformation of mammalian cell cultures, but infectious virus has not been recovered.							Agent has not been propagated in cell culture.
Genetic susceptibility of host.	Specific cellular susceptibility and resistance in vivo and in vitro, probably determined by one or another of 2 genetic loci.							Genetic factors influence susceptibility but are probably independent of those affecting response to RIF-positive viruses.
Immunology	Antigenic in chickens and other animals. Three distinct antigenic subgroups have been identified with the neutralization test. They correspond to subgroups A, B, and C as determined by the interference and the host susceptibility characteristics. Antigenic variation is usually unrelated to variation in oncogenic spectrum. There is a complement fixing antigen (COFAL) which is common to the three subgroups. Most chickens in commercial flocks have neutralizing antibody. High antibody titers will not protect against tumor formation.							No evidence of a neutralizing antibody.
Epizootiology: Distribution	Found wherever poultry is kept.							Found in most poultry areas.
Occurrence	Common, especially in commercial flocks.	Rare.	Sporadic.	Sporadic.		Sporadic.	Sporadic.	Endemic and often assumes epidemic proportions.
Mortality	Low to moderate rate over long period.	Very low.	Very low.	Very low, but rarely high.		Very low.	Very low, but occasionally high.	Moderate to high rate over short period followed by low rate over long period, or only a low rate.

THE AVIAN LEUKOSIS COMPLEX—Continued

Item	Diseases Caused by RIF-Pasitive Viruses							Diseases Caused by RIF-Negative Agents
	Lymphoid Leukosis (LL)	Myeloblastosis (Leukemie)	Myelocytomatosis (Aleukemie)	Erythroblastosis		Fibrosareoma, endothelioma, and nephroblastoma	Osteopetrosis	Marek's disease (MD) (including acute leukosis)
				Proliferative	Anemie			
Epizootiology—Con. Transmission (natural).	Carriers—common at all ages and for long periods; congenital (egg)—mueh; direct eontaet—moderate; eontaminated environment—little; airborne—little if any.	Primarily unknown; some direet eontaet transmission oecurs.						Carriers—unknown; eongenital—little if any; direet eontact—mueh; eontaminated environment—probable; airborne—mueh; highly eontagious; arthropod veetor implieated.
Suseeptible hosts	Chickens most suseeptible; oeeurs in all breeds; turkeys moderately suseeptible.	Chiekens most suseeptible. Variable experiment transmission to guinea fowl, turkeys, doves, pheasants.						Chiekens most suseeptible; deseribed in dueks, pheasants, and turkeys.
Factors influeneing suseeptibility:								
Age	Suseeptibility decreases with age.							Suseeptibility decreases with age.
Seasonal	No effeet.							No effeet.
Sex	Female more suseeptible.	Little if any sex effect.					Males more suseeptible.	Females more often affeeted than males.
Environment and other infeetions.	Unknown.							Enhaneement by other infeetions and stress suspeeted but unknown.
Symptomatology: Incubation period	5–8 months.	3–16 weeks.	Unknown.	3–16 weeks.		2–16 weeks.	3–16 weeks.	2–16 weeks.
Signs	Enlarged abdomen, palpable liver, weakness.	Paleness, weakness, emaeiation with or without enlarged abdomen or tumors.		Weakness with or without paleness and emaeiation.		Palpable tumors in musele or skin; weakness.	Abnormal (symmetrieal or irregular) enlargement of long bones.	Asymmetrie progressive paresis of leg, wing, or neek; ineoordination, emaeiation, dehydration, dyspnea; weakness, signs not always obvious. Enlargement of feather follieles, leathery skin, palpable tumors. Abnormal grayish eolor of iris; eonstrieted, irregular, or fixated iris.

THE AVIAN LEUKOSIS COMPLEX—Continued

Item	Diseases Caused by RIF-Positive Viruses							Diseases Caused by RIF-Negative Agents
	Lymphoid Leukosis (LL)	Myeloblastosis (Leukemic)	Myelocytomatosis (Aleukemic)	Erythroblastosis		Fibrosarcoma, endothelioma, and nephroblastoma	Osteopetrosis	Marek's disease (MD) (including acute leukosis)
				Proliferative	Anemic			
Pathology: Pathogenesis	All virus strains or isolates studied are multipotent, causing several types of tumors in susceptible chickens. If exposure is early and heavy, a permanent viremia (immunological tolerance) occurs and antibodies are not produced. When exposure is light or late, viremia disappears and is followed by antibody production.							Infection is soon followed by infiltration of peripheral nerves, brain, cord, oculi, and in some cases visceral organs and other tissues. As the proliferative phase progresses, birds develop clinical signs which may lead to death. In some, frank neoplasia (lymphoblasts) occurs; in others, terminal lesions are composed of inflammatory cells. Virus is demonstrable in blood and visceral organs. Observations suggest a latent infection or carrier state. Antibody has not been demonstrated. Apparent recovery from clinical signs occurs in some birds. Evidence of carrier states, congenital infection of embryos, viremias, or antibodies in such recovered birds is not available.
	Whether or not viremia is persistent or transient with antibody formation, chickens may appear normal, and histopathologic lesions may be absent for long periods. Acquired antibodies have little or no influence on tumor growth. Shedding of virus into the egg, the nasal and salivary secretions, and droppings occurs consistently in viremic chickens and sporadically if at all in chickens having antibodies.	Viremia is accompanied by proliferations of the primitive blood elements first in the bone marrow and then also in parenchymatous organs and some tissues. If the course is not rapid, antibody is formed.				Viremia is soon followed with proliferations of respective tissue elements.		
Cell types	Predominantly lymphoblasts with variable numbers of small to large lymphocytes.	Myeloblast.	Myelocyte.	Erythroblast.	Polychrome erythrocytes; few blast cells.	Fibroblast, endothelial cell, epithelial cell.	Osteoblasts.	Small, medium, and large lymphocytes, plasma cells, dark staining Marek's cells, and reticular cells; in some cases, many lymphoblasts.
Morphology (gross): Liver	Usually tumorous; diffuse or focal and enlarged 2–10×.	Usually tumorous, diffuse and moderately enlarged.	Often tumorous; usually nodular white masses; slight or no enlargement.	Usually involved, cherry to mahogany red, moderately enlarged.	Pale, not enlarged.	Occasionally focal tumors with no liver enlargement.	No change or may be fibrotic.	Seldom to often tumorous, diffuse or focal and enlarged 2–5×.
Pancreas	Seldom tumorous; some enlargement; firm, whitish and nodular tumors.	Rarely tumorus, similar to LL.	Occasionally white nodular tumors.	No changes.			No changes.	Similar to LL.
GI tract	Occasionally tumorous; usually nodular in intestinal wall, diffuse tumor in wall of proventriculus.	Occasional tumor, similar to LL.						Crop or other parts may be distended due to nerve disfunction. Proventriculus commonly diffusely tumorous, intestine occasionally tumorous, usually nodular.
Mesentery	Infrequently tumorous; diffuse or focal.							
Spleen	Usually tumorous; diffuse or focal and enlarged 2–20×.	Usually diffusely tumorous, enlarged 2–20×.	Often tumorous; usually nodular white masses; slight or no enlargement.	Usually involved; slight to moderately enlarged, cherry to dark red.			Atrophy, usually with pigmentation.	Seldom to often tumorous; diffuse or focal, enlarged 2–20×.

THE AVIAN LEUKOSIS COMPLEX—Continued

Item	Diseases Caused by RIF-Positive Viruses						Diseases Caused by RIF-Negative Agents
	Lymphoid Leukosis (LL)	Myeloblastosis (Leukemic)	Myelocytomatosis (Aleukemic)	Erythroblastosis		Fibrosarcoma, endothelioma, and nephroblastoma	Osteopetrosis	Marek's disease (MD) (including acute leukosis)
				Proliferative	Anemic			
Morphology (gross)—Con. Bursa of Fabricius	Often tumorous; diffuse or focal often with necrotic centers, enlargement moderate to great.	Occasional tumors similar to LL.	No changes.	No changes.		No changes.	No changes.	Rarely tumorous.
Thymus	Rarely tumorous; diffuse, moderately enlarged.	No changes.						Rarely tumorous, diffuse, moderate to greatly enlarged.
Gonad	Ovary occasionally tumorous, diffuse, lobulated, moderate to greatly enlarged; testis rarely tumorous.	Occasional tumor similar to LL.						Ovary commonly tumorous; diffuse, lobulated or smooth, slight to greatly enlarged. Testis often tumorous; focal or diffuse, slight to moderately enlarged.
Kidney	Frequently tumorous; usually diffuse, moderate to greatly enlarged.		Occasional white nodular tumors.	Some congestion.		Nephroblastoma—few to many, large or small, focal cystic or solid tumors.		Seldom to frequently tumorous, focal or diffuse, slight to greatly enlarged.
Heart	Seldom tumorous; diffuse or focal in myocardium, sometimes nodular tumors on epicardium and in myocardium. May be dilation or thickening of chamber walls when function is impaired.			No changes.		Occasionally may have fibrosarcoma or endothelial tumors.		Frequently tumorous; nodules on epicardium and in subepicardial fat; diffuse or focal tumors in myocardium and occasionally large nodular tumors.
Bone marrow	Usually tumorous; grayish, diffuse or nodular.	Always tumorous, gray to white, diffuse.	Myelocytic hyperplasia.	Fluid, cherry to dark red.	Aplastic.	No changes.	Reduced.	Gross alteration uncommon.
Blood	Occasional lymphocytosis or leukemia.	Large numbers of myeloblasts with few to many myelocytes.	Usually aleukemic.	Pale, watery blood; clots poorly.		Rarely anemic.	Usually anemic.	Unknown.
				May be no obvious change, variable number of erythroblasts and polychrome erythrocytes.	Many polychrome erythrocytes.			
Lungs	Rarely tumorous; usually diffuse, firm, gray, occasionally focal.	Occasional tumor similar to LL.	Occasional white nodular tumors.	No changes or congestion.		Occasionally may have fibrosarcoma or endothelial tumors.	No changes.	Commonly tumorous; diffuse, firm, yellow-gray, often edematous.
Muscle	Very rarely tumorous; diffuse or nodular.	No changes.	Yellowish-white tumors.	No changes.		Fibrosarcoma or endothelial focal tumors may occur.	Muscle atrophy of affected appendages.	Seldom to frequently tumorous; nodular or streaking between muscle bundles, gray to yellowish-white, with or without gelatinous fluid; occasional massive diffuse tumors. Atrophy of muscles which are innervated by affected nerves.

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				Proliferative	Anemic			
Morphology (gross)—Con. Skin	Very rarely focal tumors.	No changes.	No changes.	No changes.		Fibrosarcoma or endothelial tumors may occur.	No changes.	Seldom to frequently tumorous; nodular enlargement of feather follicles, dark yellow to gray; occasional general thickening and ulceration.
Bone	No changes.		Usually tumorous; white to yellow on serosal surface of ribs and other bones.			No changes.	Always involved; moderate to marked enlargement of diaphysis of long bones; also rough surface and thickening periostium.	No changes.
Peripheral nerve and ganglia.	Very rarely extension of tumor to nerves.		No changes.			Fibrosarcoma may occur.	No changes.	Usually involved; enlargement, discoloration, or loss of striations in single or several nerves and ganglia. Grossly normal nerves may have extensive microscopic cellular infiltration.
Brain and spinal cord.	Usually no gross changes; occasional involvement of meninges with extension into brain.							Usually no gross changes.
Eye	No changes.					No changes.		Rarely to often involved; uneven depigmentation of iris with irregularity of border and constriction of pupil.
Histopathology	Mostly lymphoblasts, some small to large lymphocytes in diffuse or focal accumulations in extravascular tissue areas. Often very anaplastic.	Massive extravascular accumulations of myeloblasts and immature myelocytes in bone marrow, liver, and spleen.	Compact masses of myelocytes having acidophilic granulation.	Accumulation of erythroblasts in sinusoids of bone marrow, spleen, and liver with presence of blastic cells in vessels.	Bone marrow aplastic or shows increase in polychromes.	Sarcoma—compact masses of fusiform fibroblasts giving tumor an irregular appearance. Endothelioma—proliferations of vascular endothelium into compact or cavernous masses; the latter is due to large accumulations of blood and are hemangioendotheliomas. Nephroblastoms—proliferation of endothelial and connective tissue elements of the kidney. Tumors may be solid or cystic.	Hyperplasia of periostium; deposition of fibrous, cellular, spongy bone on the surface of the cortical bone which becomes calcified, leaving irregular spaces.	Nerve.—Lesions are of 2 types: (1) proliferative—light to heavy infiltration with lymphocytes and dark staining MD cells; when extensive there may be demyelination with Schwann cell proliferation which may extend outside of the nerve; (2) edematous—edema between nerve fibers and light infiltration with small lymphocytes and plasma cells; edematous lesion is believed to follow the proliferative lesion. Viscera.—Lesions in the viscera start with perivascular infiltrations which progress to moderate or massive accumulations of mostly small to medium and few large lymphocytes. Oculi.—Perivascular infiltration of small and medium lymphocytes with variable proportion of plasma cells in the iris and often in the ciliary body and choroiden and occasionally in the optic nerve. Brain.—Accumulation of small lymphocytes around vessels in brain (especially medulla) and cord and in central white matter of cerebellum.

THE AVIAN LEUKOSIS COMPLEX—Continued

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	Lymphoid Leukosis (LL)	Myeloblastosis (Leukemic)	Myelocytomatosis (Aleukemic)	Erythroblastosis		Fibrosarcoma, endothelioma, and nephroblastoma	Osteopetrosis	Marek's disease (MD) (including acute leukosis)		
				Proliferative	Anemic					
Diagnosis: Morphologic	Cross and microscopic lesions given in foregoing sections.									
Etiologic	A positive RIF or COFAL test or presence of Rous sarcoma or leukosis virus antibody indicates infection with RIF-positive virus, but such infection may be unrelated to observed pathologic lesions. Because of the possibility of dual infection, the RIF, COFAL or antibody tests are of little or no value in distinguishing lesions of LL from those of MD in individual birds.									
Epizootiologic features useful in distinguishing LL from MD.	Sporadic occurrence after 4 months with low rate of mortality and almost complete absence of paretic signs and neural lesions.							The following epizootiologic types have been recognized: (1) In young birds usually signs of paresis, often sudden death with a high rate and definite peak in mortality, always neural lesions, often with gonad tumors and occasionally other tumors; (2) In young birds, clinical signs often absent, may have weakness or paresis, usually sudden death with high rate and definite peak in mortality, multiple tumors of the viscera especially the liver, heart, spleen, lung, kidney, gonad, and proventriculus. Some flocks have high incidences of skin or muscle lesions. (3) In mature birds, clinical signs as in (2) above, sporadic occurrence of neural or visceral lesions.		
Differential—Conditions that may result in similar gross appearance.	Visceral lesions of MD. Pullorum disease. Tuberculosis. Enterohepatitis. Hjarre's disease. Fatty degeneration of liver. Myeloblastosis. Erythroblastosis.	LL and conditions listed under LL. Erythroblastosis.	Tuberculosis. Myeloblastosis. LL. Pullorum disease.	Passive congestion due to a variety of infectious agents. Myeloblastosis.	Anemia due to nutrition, toxic agents, hemorrhagic disease.	Muscle necrosis. Granuloma. Hemorrhage. Ovarian tumor. LL.	Callus after fracture. Perosis. Thickening due to age. Osteomyelosclerosis.	Newcastle disease. Avian encephalomyelitis. Riboflavin deficiency. Staphlococcus arthritis. Infectious synovitis. Perosis. Eastern equine encephalomyelitis. Botulism. Transient paralysis (pseudobotulism).	Lymphoid leukosis. Conditions listed under LL. Carcinoma of ovary. Fibrosarcoma. Dermatitis. Bluecomb.	Genetic gray eye. Cataract due to avian encephalomyelitis virus. Salmonella and other bacterial infections.
Prognosis	Individual: Unfavorable; most cases not reversible; rarely, apparent recovery or loss of signs. Flock: Mortality usually continues at low to moderate rates for several weeks or months. No known procedures will reverse natural course of disease.							Individual: Unfavorable; most cases not reversible; occasionally, apparent recovery or loss of signs. Flock: Mortality usually continues at moderate to high rates for several weeks. No known procedures will reverse natural course of disease.		
Prophylaxis	Obtain progeny from genetically resistant chickens. Obtain progeny from infection-free breeding stock. Hatch, brood, and rear in strict isolation and in sanitized, vector-free environment. "All-in all-out" management recommended.									

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